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# **Health Investment and Long run Macroeconomic Performance: a quantile regression approach**

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## Resumo

A saúde desempenha um papel inquestionável no bem-estar dos indivíduos e desenvolvimento dos países. No que toca à sua contribuição para o crescimento económico, existem, contudo, visões divergentes. Estas visões têm por base resultados de estimações que apenas consideram a influência da saúde sobre o valor médio das taxas de crescimento do produto/rendimento das amostras analisadas, ignorando a possibilidade de efeitos diversos dos regressores para diferentes valores da variável dependente. Ou seja, ignoram que a influência da saúde pode ser heterogénea dependendo da própria evolução da economia. A análise que desenvolvemos no presente trabalho, para uma amostra máxima de 92 países respeitante ao período 1980-2010, comprova esta heterogeneidade. Com efeito, através da estimação de uma regressão de crescimento *ad hoc* onde aplicamos a metodologia de Canay (2011) para regressão por quantis (Koenker 2012a; Koenker 2012b), verificamos que variações na saúde têm um impacto positivo no crescimento económico e que esse impacto é maior para países com taxas de crescimento baixas. Estes resultados aplicam-se quando consideramos indicadores de saúde positivos (esperança média de vida, consumo de calorias diário por pessoa) e negativos (taxa de mortalidade infantil e taxa de subnutrição). Destes concluímos que cortes em saúde devem ser cuidadosamente ponderados, principalmente em alturas de abrandamento das taxas de crescimento, pois melhorias na saúde pode fomentar o crescimento económico e a sua negligencia pode ter consequências negativas sobre as taxas de crescimento de uma economia.

**Palavras-chave:** saúde, capital humano, crescimento económico, regressão por quantis

**Classificação JEL:** C31, C33, I15, O15, O47

## **Abstract**

Health plays an unquestionable role in people's welfare and the development of countries. As regards its contribution to economic growth, there are divergent views. Some views are based on estimates that only consider the impact of health on the average value of growth rates, ignoring the different effects of regressions on the different values of the dependent variable. In other words, ignoring that the influence of health can be heterogeneous depending on the evolution of the economy itself. The analysis developed in this work, for a maximum sample of 92 countries over the period 1980-2010, proves this heterogeneity. In fact, by applying an *ad hoc* growth regression where we followed the methodology of Canay (2011) for regression by quantiles (Koenker 2012a; Koenker 2012b), we found that variations in health have a positive impact on economic growth and that this impact is greater for countries with low growth rates. This results apply to either positive (life expectancy, consumption of calories per person per day) and negative (infant mortality rate, prevalence of undernourishment in populations) indicators. We thus conclude that cuts in health should be carefully balanced, particularly in times of growth slowdowns, since health increments foster growth and neglecting it endangers an economy's growth.

**Keywords:** health, human capital, economic growth, quantile regression

**JEL Classification:** C31, C33, I15, O15, O47

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### **List of Abbreviations**

ASR – Adult Survival Rates

CVA – Cerebrovascular diseases

FRED – Federal Reserve Bank of Saint Louis (2015)

GDP – Gross Domestic Product

LE – Life Expectancy

OECD – Organisation for Economic Co-operation and Development

PPP – Purchasing Power Parity

PWT – Penn World Table

USD – United States Dollar

WDI – World Bank's World Development Indicators (2014) database

## 1. Introduction

Human capital is acknowledged as one of the most important sources of economic growth. As countries move towards knowledge-based economies, the existence of highly skilled human capital becomes increasingly important. It is thus not surprising that previous empirical research has focused on identifying the mechanisms of transmission from human capital accumulation to growth, and on assessing their magnitudes (see *e.g.* Benhabib & Spiegel 1994, 2005; Bloom *et al.* 2001; Howitt 2005; Hanushek & Woessmann 2011; Bleakley 2010).

However, most of these studies focus on formal education as the main source of human capital, while the impact on growth of health human capital has been much less studied. Health helps increase labour productivity, the capacity to learn at school and to grow intellectually and physically. Simultaneously, the decrease of mortality and morbidity allows for an increase in the proportion of the working age population, therefore contributing to raise per capita income. They also mean higher longevity, which in turn creates a greater need for people to save for their retirement (Bloom & Canning 2012). It is thus not surprising that “several of the great ‘takeoffs’ in economic history [...] were supported by important breakthroughs in public health, disease control, and improved nutritional intake [...]” (Sachs 2001; p.22). A look at some recent data at the World level (source: WDI – World Development Indicators) seems also to support this positive correlation. For instance, over the period 1970-2010 life expectancy at birth increased by 10 years – from 60 to 70 years of age for the average world citizen – and infant mortality rate decreased from 97.6 to 37.5 per 1,000 births, while at the same time World GDP (Gross Domestic Product) per capita increased from 4240,72 to 7603,14 USD at 2005 PPPs (Purchasing Power Parity). The same conclusion applies to groups of countries defined according to income levels.

At the empirical level, however, Lewis and Jack (2009), p.2 “[...] caution the reader against expecting to find consensus in the empirical literature on the links from health to growth or even from health policies to health”, highlighting the need to adopt econometric methodologies that deal with issues such as endogeneity and measurement error. As far as econometric methodologies for the study of the relationship between health human capital and economic growth are concerned, the empirical growth literature also usually assumes homogeneity among economic growth models by restricting the significance and magnitude

of the economic relations for the average country. However, it is unlikely that the impact of human capital on economic growth is the same, for instance, in the United States and in African countries. In addition, since many papers concentrate on specific countries, worldwide differences in the linkages are not adequately taken into account. Allowing for parameter heterogeneity in the study of the relationship between health human capital and economic growth in broad samples of countries can thus bring new insights. This is particularly true at a time when many countries are facing a period of crisis, with impressive growth slowdowns that demand a rigorous identification of the most effective sources of growth in periods of deceleration. For instance, in 2007 Angola recorded an output growth rate of 4.97% and the Euro Area a growth rate of 19.02%, but in the period following the financial crisis – 2008-2014 – the average growth rates declined to 1.74% and 1.75%, respectively (source: FRED). The identification of different impacts on growth of health human capital across the growth rate distribution can be especially important for countries facing fiscal sustainability issues, such as Portugal. In fact, investments in health are mainly publicly funded and a cut in public health expenditures, with the associated negative impact on health human capital, can be especially harmful for the output growth of under-performers.

The main aim of this paper is to empirically assess the importance of health human capital for economic growth in a broad sample of countries, explicitly taking into account the issue of parameter heterogeneity. For this purpose, we allow for differentiated effects of health human capital on the output growth rate, conditioned by the location of the dependent variable at different parts of its distribution. Additionally, we will analyse the sensitivity of our results to the use of different proxies for health human capital, trying in this way to overcome, to some extent, the measurement error problem.

This paper applies a quantile regression approach to estimate output growth equations in order to account for parameter heterogeneity in the relationship between health human capital and growth in a sample of 60 countries over the period 1980-2010. Quantile regressions are used as a method to capture parameter heterogeneity across countries considering varied effects across different quantiles of the distribution of the dependent variable. The data used comes mainly from the Penn World Table 8.0, World Development Indicators, CANA and Barro and Lee data set.



The remainder of this paper is organized as follows: in section 2 we review the theoretical predictions and empirical evidence on the nexus between health human capital and economic growth. Section 3 contains descriptive statistics and analyses the main variables for our empirical study. In section 4 we present the empirical model and describe the methodological approach. In section 5 we present and discuss the results. Finally, section 6 contains the main findings and some suggestions for future research.

## **2. Health as a determinant of Economic Growth: Theory and Evidence**

Economic growth research is mainly concerned with identifying those economic structural characteristics that can explain differences in standards of living and income growth across countries. Since at least Adam Smith (18<sup>th</sup> century), the study of the effects of human beings on the behaviour of aggregate output has been a matter of debate (Savvides & Stengos 2009). Nonetheless, for a long time the focus was on physical capital as the main source of economic growth. The debate was reignited by the work on the economic role of human capital by Theodore Schultz (1960;1961) and Gary Becker (1964)<sup>1</sup> during the second half of the twentieth century. As a result, human capital became widely recognized as an important source of economic growth.

This link between human capital and economic growth has been explored and measured mainly considering formal education (Mincer 1981; Benhabib & Spiegel 1994; Miles 2004). During the 20th century, health improvements were impressive. It is thus not surprising that pioneer work concerned with understanding the role of nutrition (Fogel 1994) and longevity (Preston 1975) in explaining income variations across countries was developed. Investments in health showed an upward trend, with health expenditure per capita at the World level in 2011 international dollars (PPP) rising from 481 (1995) to 605.8 (2000), and World health expenditure in percentage of GDP increasing from 8.7 (1995) to 9.1 (2000) percent (source: WDI). As one might expect, these developments resulted in extending the concept of human capital to include, besides education, “the general state of health of the working population” (Savvides & Stengos 2009), which has become popular among

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<sup>1</sup> These authors model education as an investment in human capital and highlight its importance to explain income growth at the individual/worker level. Schultz (1960), p. 572 considered that “a serious fault in the way capital is treated in economic analysis has been the omission of human capital”. See also Barro (1991) and Mankiw Romer and Weil (1992) that showed its importance at the macroeconomic level based on a theoretical growth model.

researchers, notwithstanding the divergences at the empirical level on how to measure this concept.

It is important to notice, however, that the relationship between health and economic performance can be studied from two perspectives: the macroeconomic one, which considers the economic benefits of health for the aggregate economy, and the microeconomic one, that is concerned with the individual economic benefits of health human capital (see Lewis & Jack 2009). The first perspective is the one most closely related to growth theory and will thus be the focus of our analysis.

## **2.1. Theoretical predictions on the nexus between health human capital and economic growth**

The benchmark for modelling the relationship between health human capital and economic growth is the neoclassical growth model, which has its genesis in the work developed by Solow (1956) and Swan (1956). Mankiw Romer and Weil (1992) emphasized the role of human capital in explaining income and growth differences across countries, set out in what became known as the Augmented Solow Model.

Health, just like education, differs across individuals and consists of a stock that can depreciate or increase over time (Grossman 1972). Healthier workers are able to think better, are more focused, and allocate more energy and higher effort to task performance. It is therefore possible to say that health defines some of those workers' qualitative characteristics that allow them to be more or less productive. Human capital in turn is defined as the characteristics that make a worker more productive as a result of a previous cost (Becker 1964; Filer *et al.* 1996); thus, better health results into higher human capital. Additionally, healthier workers are less likely to miss work due to sickness (Bloom & Canning 2000). The better is health for the same amount/number of labour/workers, the higher is their productivity and the output. Furthermore, if working-age population lives longer, more output can be produced, representing higher macroeconomic benefits from human capital health investment.

As a consequence of the previous view, health is considered in neoclassical growth models as just another input into the production of final goods alongside physical and education human capital. In the light of the neoclassical growth theory, higher rates of accumulation of both human (health and education) and physical capital lead to permanently higher levels of income. However, since there are decreasing returns to capital, there will

only be transitory growth effects (see Weil 2005). In the long run, income per capita grows at the same growth rate as technological progress, assumed constant and exogenous, the main driver of economic growth. In any case, poorer countries are predicted to grow at a faster pace in the neighbourhood of the steady state growth equilibrium, after differences in structural characteristics across countries are controlled (Barro & Sala-i-Martin, 2004).

Besides its role as an input into final goods production, health human capital can also play a role as an input into innovation and imitation activities. Endogenous growth theories that developed from the mid-1980s onward aimed at explaining how technological progress takes place (Jones & Vollrath 2013). AK-type growth models assume that human capital contributes to economic growth because workers with higher human capital levels increase not only their own productivity but also that of other individuals with whom they perform different tasks, thus overcoming the growth effects of the diminishing marginal returns hypothesis (Lucas 1988).

Human capital is also viewed by endogenous growth theory as being of major importance for innovation and technology diffusion activities that result from intentional decision by economic agents (Romer 1990; Nelson & Phelps 1966; Barro & Sala-i-Martin 2004). Countries differ in their production capacity and ability to improve it, with some countries relying on their ability to innovate and create new technologies (leader countries) and others on their ability to imitate those new technologies (followers). Human capital has the potential to influence both activities. According to this view, if health human capital increases, the knowledge inducing the production of new ideas/technologies will also rise, there will be more ideas available and thus more innovation will take place in the technological leader countries. In the follower countries, a better health status will increase their absorption capacity in terms of adapting and implementing the technologies developed by the leaders.

Additionally, health human capital can produce an indirect growth impact through its influence over other growth determinants such as demography, education, physical capital, and income inequality and poverty (Bloom & Canning 2000; Howitt 2005; López-Casasnovas *et al.* 2005).

Mortality rates and life expectancy affect the demographic structure of a country. Higher life expectancy and lower mortality rates imply a greater number of individuals of working age that can thus contribute to higher output levels. Additionally, if they also imply

a greater proportion of the working age population it seems reasonable to expect that output per capita will also be higher.

If the health status of the population increases, school absence due to sickness is expected to decrease. In the same way, health allows to enhance learning capacity, since individuals will be better prepared to learn both physically and intellectually. In particular, better nourished children will have better cognitive skills (Alderman *et al.* 2006). Moreover, if health increases occur in the form of decreasing mortality or increasing longevity, the higher will be the incentive to invest in education and acquire additional school qualifications. Since education is a source of human capital that is predicted to impact growth positively, healthier populations will also present higher educational attainment levels and perform better at school, which in turn leads to higher growth and income levels. Is it thus not surprising that several studies have considered the health-education nexus when studying the impact of the former on economic growth (Kremer & Miguel 2001).

Moreover, increasing longevity influences savings decisions. If people expect to live longer, they will save more for their retirement. Higher savings rates will in principle lead to higher investment rates and thus more physical capital accumulation, which in turn fosters growth in the medium term (see Barro & Sala-i-Martin 2004). On the contrary, if people's health is poorer and they have "a short time horizon because they expect to die young, they have less reason to save and the economy fails to grow." (Lorentzen *et al.* 2008, p.82).

Finally, promoting health can not only spur economic growth and development but also reduce poverty (Sachs 2001). In fact, health improvements have larger impacts on the standards of living of the less privileged people with poorer health (Deaton 2003). Poorer population which are better nourished see their education capabilities improve (Lorentzen *et al.* 2008) with positive consequences on their performance and economic growth. This is often the reason why improving the health status of the poorer is seen as a way to escape from poverty traps (Sala-i-Martin 2005).

As a final remark, notice that health differentiates itself from other economic growth sources due to its role at the social level that makes it a very sensitive area of intervention for both policy makers and individuals. We acknowledge that investments in health are fundamental at the social and humanitarian level, besides their role on economic performance.

## 2.2. Empirics

At the empirical level researchers have yet to reach a consensus on the impact of health status and accumulation on growth. Most previous empirical works find a positive relationship between health and economic growth, supporting the pioneer work of Preston (1975)<sup>2</sup>. Likewise, there is evidence pointing to health as an important growth determinant regardless of the period under analysis, type and number of countries included in the sample, health proxies used, and model specification (control variables included; ways of introducing health variables). However, negative and statistically significant impacts of health on output growth are also found (see Lewis & Jack 2009). There is also the possibility that the health proxy considered accounts for mismeasured/not included factors, as first suggested by Barro & Sala-i-Martin (1995). This has led economists to search for patterns across specific regions and among countries within the same group of income level (Eggoh *et al.* 2015; Poças & Soukiazis 2012; Aghion *et al.* 2011; Bhargava *et al.* 2001). Nevertheless, estimation results seem sensitive to the health proxies used and the health interactions considered. The following subsections provide a more detailed discussion on the matter. Table A.1. in annex A provides a summary of the empirical studies reviewed in this section.

As far as panel data studies with wide samples of countries are concerned, Bloom *et al.* (2004) review some previous studies that use life expectancy to proxy health and conclude from this literature review that the majority find a positive effect running from the initial level of health to output growth. They also estimate an economic growth regression, with life expectancy (initial level) as the main explanatory variable, over the period 1960-1990 and with a sample of 104 countries. They start with an aggregate production function that takes into account differences in productivity among countries. The results found point to statistically significant and positive coefficients for life expectancy, suggesting that health affects economic growth through its direct impact on labour productivity.

Also using life expectancy to measure health human capital accumulation, Acemoglu & Johnson (2007) arrive at a negative coefficient for a sample of 47 countries over the period 1940-1980 implying that faster health accumulation is not beneficial for economic growth. The authors explain these results on the basis of a Malthusian effect (the idea that population growth is expected to exceed resources growth) since for the period

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<sup>2</sup> Preston's (1975) work finds a strong positive correlation between life expectancy and income.

under analysis life expectancy grew at the same rate as the population. In line with this work, Aghion *et al.* (2011) mainly aim at proving that, even when using different instruments for health, negative versus positive coefficients are due to a missing effect: health in some countries grows faster because they have lower initial levels of health (health convergence). They estimate a cross country regression for 96 countries where they find a positive impact going from health to growth although health growth/accumulation reveals to be less robust<sup>3</sup> (beyond a certain point health growth becomes statistically insignificant). They also instrument life expectancy with the same instrument variables as in Lorentzen *et al.* (2008), whose focus is on the relationship between life expectancy and adult survival rates<sup>4</sup>, but the results remained almost the same. Additionally, for the same time span Lorentzen *et al.* (2008) explore other channels of interest through which health might influence growth; they conclude that, when investing in physical capital and influencing fertility rates, health can affect growth in a quantitatively more important way than through human capital investments. Aghion *et al.* (2011) follow their lead and also extend their work to ascertain these mechanisms, although in their work both the initial level and the growth rate of health influence the level of fertility whereas health impacts investment in human and physical capital.

Cooray (2013) and Soukiazis & Cravo (2007) try to disentangle in a different way the relationship between health and economic growth by grouping the sample according to countries' income levels. They also use health in the form of life expectancy, but Cooray (2013) uses a sample of 210 countries and Soukiazis & Cravo (2007) 77 countries (for the periods 1980-2000 and 1990-2008, respectively). Despite using the same health proxy, Cooray (2013) finds correlation between health and other variables such as health expenditure and education while Soukiazis & Cravo (2007) do not. Increasing health, according to the results in Soukiazis & Cravo (2007), is growth enhancing for low income countries whereas it has no statistically significant impact in high income countries. When Cooray (2013) uses adult survival rates as a proxy for health, the results found point to a positive growth influence in upper middle and high income countries while the influence is negative in low and low middle income countries.

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<sup>3</sup> Results only of the Bayesian approach also applied.

<sup>4</sup> Lorentzen *et al.* (2008) use an instrumental variable approach, more specifically they regress economic growth with life expectancy and use adult survival rates as instrumental variables.

The study by Bhargava *et al.* (2001) measures health as adult survival rates considering a sample of 92 countries and obtains results similar to those of Soukiazis & Cravo (2007). Their results, notwithstanding, indicate that the impact of health on growth is significant until it reaches a certain threshold, above which it becomes insignificant, providing a possible and reasonable explanation for the differences in the estimated results of other studies.

Similarly to some previous studies, Cooray (2013) also explores the interrelations (with interaction terms) between health and other variables that might influence economic growth. The author finds statistically significant and positive effects of health on education human capital (in line with results of the previous studies of their review) and health expenditure.

In addition, Wang (2011) investigates the impact of health expenditures on growth by estimating a growth regression for 31 countries over the period 1986-2007. The author examines the health-growth link from a different perspective to provide a complete picture of the influence of health on economic growth in what concerns the existence of non-linearities. In particular, he applies a quantile regression approach to identify different health expenditures growth impacts at different parts of the output growth rates distribution. The results obtained indicate that there is a positive influence among middle and high performers in terms of output growth, while for low performers the influence is negative.

By contrast, some researchers narrow the sample to specific countries within a geographical region or an institutional group, trying in this way to have a deeper understanding of the health contribution to economic growth (*e.g.* Eggoh *et al.* (2015) for African countries and Poças & Soukiazis (2012) for OECD countries). They both try for the use of several variables to quantify health (see table A.1.). Poças & Soukiazis (2012) find evidence that health boosts growth in OECD countries, especially when measuring the dimension of health care quality and the mortality rates of specific diseases. On the other side, Eggoh *et al.* (2015) inference that increasing health expenditure may have a negative influence on growth, even though the level of health expenditure for the countries is low, when education expenditures are below a certain threshold.

A matter of great importance concerns the ability to statistically summarize all the information of a particular relation between variables using usual estimation techniques. In general, when addressing health impact on economic growth, linear regression estimates the

average effect of health on output growth rates. In this way it is possible to assess if health is important for economic growth. On the other hand, it cannot provide information on whether health influence on economic growth differs for under-performers more than for average performers. Soukiazis & Cravo (2007), Bhargava *et al.* (2001) and Cooray (2013) try to go beyond the usual thinking and figure that by dividing their sample into three different groups according to their income levels (low, middle and high income countries) they could look for differences in the effects covariates could have on the dependent variable. The main goal of this work is to give a more comprehensive picture of the effects of health on growth on the basis of differentiated impacts according to the distribution of output growth rates. This is achieved by using an appropriate estimation methodology – quantile regression approach – that makes it possible to model the relation between a set of variables and a quantile of the dependent variable and thereby account for heterogeneous effects of the covariates. The effect of health on output growth rate can then be compared with its effect on the median or other quantiles of the output growth rate. Indeed, the coefficients obtained through quantile regression estimation are comparable to conventional linear ones.

Despite the theoretical arguments in favour of a positive and important influence of health on economic growth, in the empirical literature there are still some gaps to fill in order to get a better understanding of the role of health on long run economic growth rates. The main aim of this work is to contribute to shed additional light on the understanding of the health-growth nexus by applying quantile regressions in order to seek for new potential implications from the results obtained. Not only do we want to allow for a more complete view of health influence over economic growth rates using this methodology, we also want, by considering different health proxies, to contribute to develop new insights and some consensus as well as to stimulate future research on the topic. The most commonly used proxy for the health status of the population is life expectancy. In this study, although data availability did not allow us to consider as many dimensions of health as we had initially planned, we are still able to capture the two dimensions: health status and health investment.

### **3. Data overview**

Our broader sample includes balanced panel data<sup>5</sup> for 92 countries (see in annex B table B.1) from 1980 to 2010. The data needed for the estimation of our growth regressions

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<sup>5</sup> In order to have a balanced panel we applied some valid data manipulation procedures detailed in Appendix I.



were computed with information obtained mainly from the *Penn World Table* (PWT) (Feenstra *et al.* 2015), version 8.0, and the *World Development Indicators* (WDI). We chose these two databases due to the need to ensure international comparability of the data for a large sample of countries, including data for health human capital and for standard growth determinants identified in previous empirical and theoretical growth studies.

Using data originally from the PWT 8.0 we computed data for real GDP per capita at constant PPP by dividing output at constant international PPP at 2005 prices by total population. From the WDI we extracted several health proxies based on the following criteria: firstly, the availability of data for long periods of time and relative to a high number of countries; and secondly, our interest in capturing different growth effects through several perspectives of health.

Although health status and changes of a population are difficult to measure, the classical proceeding for health's evaluation is based on 5 D's: death, disease, disability, discomfort, and dissatisfaction (Lohr 1988). These provide negative outcome indicators and therefore the doubt remains whether it is more accurate and conceptually correct to measure the lack of health (negative indicators) rather than its existence (positive indicators). The latter refers to wellness and quality of life, which involves a lot of subjectivity in its measurement. Thus, besides the criteria for selecting health variables previously described, we also considered both positive and negative health indicators. We are thus able to account for these two dimensions of health status. The positive health indicators used are life expectancy, female and male survival rates to the age of 65, and consumption of kilocalories per day per person. The negative health indicators are female and male adult mortality rates, infant mortality rate and the prevalence of undernourishment. Finally, we also proxy for health investments using health expenditure per capita. For a summary of the variables gathered in our database see Table B.2. in annex B.

In order to assemble our data set, we started by considering all the countries for which there was information in the two main databases, the PWT and the WDI. We next excluded some observations based on the following criteria: first, we excluded from the sample current members of the Organization of the Petroleum Exporting Countries (OPEC) and small countries<sup>6</sup>. Standard growth determinants are probably not able to explain the

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<sup>6</sup> Countries with population lower than one million according to WDI 2014 data.

reality of those countries with their respective growth performances explained by very specific factors related to oil production in the first case and scale in the second. Second, we also dropped from the data set countries with insufficient information regarding the key variables for this study and whose real output observations started only after the year 1985. Finally, for each health indicator we adjusted the period and number of countries according to data availability and used index conciliation as well as other manipulation techniques<sup>7</sup> to fill the gaps of all the variables included in our data set.

As we can see in figure B.1 in annex, that contains an overview of the output per capita growth density function conditioned by the time period, the estimated density function of average growth rates over time shows a peak. This indicates a concentration of the probability mass, suggesting that the observed growth rates are likely to be close to the output growth rate values associated with the peak. Table 1 presents a brief summary of the main variables' data distribution based on the respective descriptive statistics. According to the information presented in Table 1, we confirm a leptokurtic shape ( $kurtosis > 3$ ) related to the unconditional distribution of output growth rates, indicating also a high concentration of values and a low standard deviation. Nevertheless, it is important to highlight figure B.2 in annex that presents a scatter plot relating the countries' average growth rates for two sub-periods 1980-1995 and 1995-2010. Although most of the dots are in the first quadrant, explaining the huge concentration of observations previously mentioned, there are considerable observations in the other three quadrants. This implies that some of the countries in our sample have registered very different figures for the respective average growth rates over that time span. This observation supports the importance to further investigate whether the explanatory variables we considered in our growth regressions have different growth impacts across different parts of the distribution of the output growth rate, our dependent variable.

Regarding health indicators, health expenditures, adult mortality rates (female and male) and the consumption of calories also present a high concentration of values with a skew to the left (higher in health expenditure) noting that the probabilistic mass concentration is on the left of the mean; this leads us to think that the main observations do not reflect the mean observations, as we pretend to ascertain in this work. With regard to the

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<sup>7</sup> Our data manipulation involves autoregressive processes fitted to the data (for more details see appendix I).

other health indicators (table 1), they seem to present a flatter distribution (level of kurtosis under 3), indicating that there are differences in the values of those indicators across countries or within countries over time, reflected in longer tails, and therefore less concentration of the data near specific values. In what concerns the distribution of adult survival rates, it appears to be similar for both genders. Regarding adult mortality rates, the respective distribution seems to have a higher concentration of values of female rates on the left side as compared to the distribution of male rates.

Since one of the objectives of this work is to assess the sensitivity of health growth influence according to the different health proxies used, it is relevant to understand how they relate to each other. Figure B.3. in annex B presents scatterplots of the relationships between health indicators of our data sets, and table B.3. in annex their correlation matrix. The negative indicators (such as mortality rates and population undernourished) are negatively

**Table 1. Descriptive statistics of the main variables**

Variable	Obs.	Mean	Stand. Deviation	Median	1 <sup>st</sup> quantile (0.25)	3 <sup>rd</sup> quantile (0.75)	Skewness	Kurtosis <sup>8</sup>
<b><i>Δlny</i></b>	552	0.0166	0.0392	0.0175	-0.0043	0.0369	0.1015	10.5891
<b><i>lile</i></b>	552	64.77	11.1228	67.94	56.08	74.11	-0.5758	2.3986
<b><i>asr.m</i></b>	552	0.6095	0.1618	0.6333	0.4864	0.7448	-0.4326	2.2845
<b><i>asr.f</i></b>	552	0.6969	0.1785	0.7478	0.5577	0.8520	-0.6119	2.2824
<b><i>imr</i></b>	534	0.0494	0.0419	0.0371	0.0114	0.0798	0.8106	2.7536
<b><i>amr.m</i></b>	540	0.2595	0.1240	0.2376	0.1629	0.3237	1.0095	3.8992
<b><i>amr.f</i></b>	540	0.1926	0.1302	0.1491	0.0886	0.2689	1.2343	4.3889
<b><i>ilgh</i></b>	270	820.93	1062.645	343.13	90.32	1248.04	1.9133	7.3132
<b><i>under</i></b>	252	0.2111	0.1331	0.1945	0.1003	0.3050	0.5538	2.6678
<b><i>ilkal</i></b>	252	154.400	105.1652	142.344	70.125	229.000	0.7449	3.3587

Source: authors' edit.

Notes: *Δlny* - real GDP per capital average growth rate; *lile* - life expectancy; *asr.m* - initial level of adult male survival rate; *asr.f* - initial level of adult female survival rate; *imr* - initial level of infant mortality rate; *amr.f* - initial level of adult female survival rate; *amr.m* - initial level of adult male survival rate; *ilgh* - initial level of public health expenditure per capita; *under* - initial level of prevalence of undernourishment; *ilkal* - initial level of consumption of calories per day per person. *Δlny*, *lile*, *asr.m*, *asr.f*, *imr*, *amr.m*, *amr.f* relate to 1980-2010. *ilgh* relate to 1995-2010 and *under* and *ikcal* relate to 1990-2010.

<sup>8</sup> Relates to Pearson's measure of kurtosis. The levels of kurtosis under 3 correspond to leptokurtic shape distribution, above 3 correspond to leptokurtic shape distributions and equal 3 present a mesokurtic distribution just like the normal distribution.

correlated to the positive ones (life expectancy, survival rates, consumption of calories), as expected. Although the correlation between health expenditure and the other health indicators does not seem to be linear, as opposite to what we see for the other correlations, it appears to present a positive sign relative to the positive indicators and a negative sign when correlated with negative indicators, as expected. In terms of the possible influence of health on economic growth, we expect that negative health indicators have a negative effect on economic growth, while positive health indicators should influence growth positively.

#### 4. Empirical growth models and quantile regressions

In order to assess the importance of health and its different proxies for long run growth we estimate what is known in the literature as an *ad hoc* growth regression (Barro & Sala-i-Martin 2004) since it is not directly derived from a particular growth model but incorporates growth determinants highlighted by both the exogenous and the endogenous growth literature. We consider each health proxy in turns (to avoid collinearity), alongside a set of control variables factors identified as important growth determinants in the empirical and theoretical economic growth literature (see annex A).

Our baseline growth regression is given by equation (1):

$$\frac{\Delta \ln y_{it}}{T} = \beta_0 + \beta_1 h_{it-1} + \beta'_{[2...p]} X_{it} + \epsilon_{it} \quad (1)$$

where  $\frac{\Delta \ln y_{it}}{T}$ , the dependent variable, is the real GDP per capita annual average growth rate for each 5-year period;  $h_{it-1}$ , the main explanatory variable, is the proxy for health given by the initial level of the variable for each 5-year period; the vector  $X$  contains the control variables identified according to previous theoretical and empirical literature -  $educ_{it}$  is educational human capital proxied by Barro and Lee (2010) average years of total schooling;  $ly_{it-1}$  is the log of initial real GDP per capita (for each 5-year period) that controls for the existence of convergence among the countries in our sample;  $gfcf_{it}$  is the share of fixed capital formation in GDP;  $n_{it}$  is the average population growth for the 5-year interval;  $g_{it}$  is average government consumption share in output for 5-year period and  $lopen_{it}$  is the logarithm of trade share in output (see annex B table B.2).  $\beta_0$  is a constant term and  $\epsilon_{it}$  the error term with the usual properties.

We assume that better health status plays a key role in fostering workers' productivity. Thereby, real income per capita rates are thought to change in line with life

expectancy, survival to age of 65, kilocalories per day and health expenditure per capita; and to vary inversely with adult mortality rates and infant mortality rates and the prevalence of undernourishment. Increasing production capacity by investment in physical capital or in education represents the possibility of increasing the amount of output, and therefore these investments also play an important role in explaining differences in growth rates across countries. But not only capital influences countries' growth rates, the initial level (supported by neoclassical approach) of output of a country will affect their performance as well. Lower growth rates are associated with countries with levels of output close to their equilibrium, whilst higher growth rates are assumed for countries far from their potential output. Population growth rates are also considered in the neoclassical framework and are used as a proxy to increments on the labour force. According to the classical hypothesis, in equilibrium, countries grow at a constant rate that depends on population growth. Besides this, a country's GDP per capita rises whenever countries expand their market to foreign countries and allow for more products to be traded with the rest of the world, and therefore the outlook for raising GDP per capita increases in the long run. Finally, Barro (1990) considers the share of government expenditures as a powerful determinant of growth rates. He argues that increasing the share of non-productive government expenditures, where the inexistence of resulting impacts on productivity is implicit, can lower growth by lowering saving rates.

Taking the standard deviation (see table 1) of life expectancy, it is expected that we come across huge differences among some countries of the sample, namely regarding public health expenditure per capita and calories consumption per day per person. For example, life expectancy at birth in Cambodia in the beginning of the period 1980-1985 was around 28 years whereas, in the same period, it was near 76 years in Sweden. Perhaps if both countries increase life expectancy in the same amount, the gains in terms of output growth arising from that increase are lower for Sweden and the effort made towards it is higher. We thus assume it has decreasing marginal returns, the same being applied to health expenditure and calories consumed per day per person. For this reason, we model differently these three variables:  $1/h_{it-1}$ . However, this reasoning does not apply to mortality rates because it is assumed that higher mortality has a detrimental impact on output growth since it means poorer health. Additionally, we opt for long run specification as a 5-year interval sample and thereby try to empirically overcome the endogeneity between output growth and health. We also benefit

from using panel data, that permits to control health variables' measurement errors (Schultz, 1994).

We have estimated equation (1) using quantile regression aiming at identifying health effects beyond those allowed by conventional procedures. This method, first proposed by Koenker & Bassett (1978), estimates models for conditional quantile functions,  $Q_\tau(Y/X)$ . This means that the influence of a set of variables  $X$  on  $Y$  is estimated for univariate quantiles  $\tau \in (0,1)$  of the distribution of  $Y$  rather than focusing on the expectation of the response variable as do least squares estimation. The mean effects reflect only a specific part of the distribution (central), and similarly univariate quantiles of the empirical distribution also correspond to a particular location of the distribution with value  $y$  such that  $P(Y \leq y) = \tau$ . So, thinking of quantiles as a central part of a particular location of the distribution (like median and mean are) makes it possible to solve the minimization problem in the same way as the conditional mean<sup>9</sup>.

In summary, the estimators of quantile regression minimize a weighted absolute sum of deviations given by:

$$\hat{\beta}_\tau = \arg \min \left[ (1 - \tau) \sum_{i \in \{i: y_{it} < X'_{it}\beta_\tau\}} (y_{it} - X'_{it}\beta_\tau) + \tau \sum_{i \in \{i: y_{it} \geq X'_{it}\beta_\tau\}} (y_{it} - X'_{it}\beta_\tau) \right] \quad (2)$$

By applying the quantile regression procedure, it is possible to generate estimates of the influence of the covariates on the dependent variable for each quantile  $\tau$  of the distribution of the response variable. Due to the complexity in applying this procedure it is computed with the linear programming procedure available in R studio (Koenker 2012b).

The quantile regression approach presents several advantages when compared to conventional estimation methods such as OLS. The most obvious one refers to the fact that it provides summary statistics on both central and tail distribution of the response variable allowing for a more complete investigation of the influence of specific covariates<sup>10</sup>. Quantile regression is also a more robust estimation procedure when the errors are not independently and identically distributed. It is also more robust to outliers since the estimators minimize asymmetrically absolute deviations. Furthermore, we can easily compare regression coefficients of specific quantiles to least squares estimates. The interpretation is very similar:

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<sup>9</sup> For the median ( $\tau = 0.5$ ) the problem is solved minimizing the absolute sum of deviations.

<sup>10</sup> The quantile regression produces similar results to conditional mean estimation when the model fits the classical linear hypothesis.

a one-unit increase in the predictor variable associated to the estimated coefficient produces a change in the dependent variable expressed by the coefficient obtained for the specific quantile of the response variable.

Furthermore, longitudinal data enables us to control for unobserved fixed effects. To address the overparametrization resulting from the parameter heterogeneity (Koenker 2004) we eliminate the fixed effects by the method proposed by Canay (2011). This consists of a two-step estimator that is consistent and asymptotically normal as both the number of individuals and the time span grow. Assuming that fixed effects affect all quantiles in the same way, the effect on the conditional mean will also be the same. Therefore, in a first step, we estimate the conditional mean within the model and then purge this model from the individual effects. In a second step, it is thus possible to run a simple quantile regression after subtracting the individual effects from the dependent variable.

Most studies previously reviewed either in this work and in Bloom *et al.* (2004) seek to identify health status effects on economic performance but fail to account for the entire conditional distribution of the output growth rate. Wang (2011) and Miles (2004) are examples of studies that investigate heterogeneous effects of different growth determinants. Miles (2004) focus on educational human capital neglecting the role of health. This author considers a sample of 77 countries for the period 1970-1998 and applies a pooled quantile regression approach<sup>11</sup>. This study finds different marginal effects of human capital between slow growers and fast growers. Wang (2011) using the same quantile approach finds differences in the effect of health care expenditures across the distribution of the output growth rate. More precisely, for higher growers he finds health expenditure growth enhancing whilst it has the opposite influence on slow growers.

In our analysis, we address our main purpose estimating firstly a *within* panel model for a better discussion and comparison of the estimation results with those obtained when applying the fixed effects quantile regression proposed by Canay (2011) (for 0.05, 0.25, 0.5, 0.75 and 0.95 quantiles). In the next section we present and discuss the results obtained.

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<sup>11</sup> Thus different from our approach.

## 5. Estimation Results

In this section we present the results of estimating our baseline growth regression with the different health proxies for our sample of a maximum of 92 countries over the period 1980-2010. Before estimating our different growth regressions, we applied unit root tests<sup>12</sup> to all variables and residuals of the regressions and all proved to be stationary<sup>13</sup>. This way, we eliminate the possibility of spurious estimations. To estimate the least squares model we thought relevant to apply the Hausman (1978) test<sup>14</sup> in order to confirm fixed effects consistency that otherwise would jeopardize the panel methodology from Canay (2011). The results from these preliminary tests allowed us to proceed to the estimation of our growth regressions with quantile techniques.

As far as the results from the quantile regression estimations are concerned, we present them in two different ways in order to facilitate the interpretation of our findings. On the one hand we plot the evolution of the marginal effects of the different health proxies across quantiles, at a 90% confidence intervals, together with the marginal effect of the least squares estimation also at 90% confidence intervals. Additionally, for each variable we present the results of the test of coefficient homogeneity<sup>15</sup> across quantiles along the output growth rate conditional distribution. The null hypothesis indicates equality of slopes across quantiles, so if the test rejects the null hypothesis we are in the presence of significant differences in slope coefficients for the explanatory variables. Furthermore, to allow for a clearer interpretation of the graphical analysis, table 2 provides an overview of the health proxies' estimated coefficients for positive indicators and health expenditure. For the estimation, we considered five  $\tau$ 's ( $\tau = 0.05, 0.25, 0.5, 0.75, 0.95$ ) because we considered that in this way we can better understand the dynamics of the changes in coefficients among the conditioned distribution.

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<sup>12</sup> For this purpose we apply the Levin-Lin-Chu (2002) test in GRETl (see appendix II for details).

<sup>13</sup> Aside from prevalence of undernourishment, calories intake and health expenditure that we were not able to perform the test.

<sup>14</sup> This test evaluates the consistency between the fixed effects estimator and the random effects estimator. We use the method *swar*, *amemiya*, *walhus* and *kinla* to estimate the random panel model and the output obtained was the same (rejecting the null hypothesis of fixed effects inefficiency).

<sup>15</sup> The test is the Wald test for marginal effects equality (Koenker & Bassett 1982).



Figure 1 plots the estimated coefficients for the different health proxies across output growth quantiles. As we can see in figure 1, part (a), the inverse of the life expectancy logarithm (*ilie*) shows a negative coefficient, as expected, that decreases from low (slow

**Table 2. Estimates of the quantile panel model and fixed effects model for positive health indicators and health expenditure**

		<i>ilie</i>	<i>ilkal</i>	<i>asr.m</i>	<i>asr.f</i>	<i>ilgh</i>
<b>FELS</b>		-1.3308*** (0.3532)	-0.0333 (0.0981)	-0.8320*** (0.1370)	0.1359 (0.0878)	-0.0566 (0.0915)
<b>Quantile</b>	$\tau = 0.05$	-1.7878*** (0.3737)	-0.0432 (0.0315)	0.0087 (0.0700)	0.1458* (0.0633)	-0.8442*** (0.1139)
	$\tau = 0.25$	-1.9006*** (0.3693)	-0.0628* (0.0277)	-0.0176 (0.0385)	0.1675*** (0.0362)	-0.7918*** (0.0775)
	$\tau = 0.50$	-1.2211*** (0.3199)	-0.05219. (0.0281)	-0.0410 (0.0417)	0.1373** (0.0457)	-0.7583*** (0.0568)
	$\tau = 0.75$	-0.9630** (0.3047)	-0.0688* (0.0343)	-0.0924* (0.0392)	0.1717*** (0.0411)	-0.8599*** (0.0662)
	$\tau = 0.95$	-0.8711 (0.7011)	-0.0383 (0.0417)	0.0158 (0.0817)	0.0646 (0.0926)	-0.8858*** (0.1465)
<b>Equality slope test</b>		2.9263 (0.0198)*	1.5782 (0.1773)	0.9327 (0.4440)	1.6671 (0.1548)	1.2765 (0.2772)
No. countries		92	63	92	92	90
Time period		1980-2010	1990-2010	1980-2010	1980-2010	1995-2010

Source: Authors' calculations in R software and edit.

Notes: *lie* - life expectancy; *asr.m* - initial level of adult male survival rate; *asr.f* - initial level of adult female survival rate; *ilgh* - initial level of public health expenditure per capita; *ilkal* – initial level of consumption of calories per day per person. Standard errors in parenthesis. For equality slope test is presented the test's statistic and in parenthesis the p-value. \*\*\*, \*\*, \* and '.' denote the statistical significance at the 0.1%, 1%, 5% and 10% levels, respectively.

growers) to high (fast growers) deciles. These findings suggest that an increase in life expectancy, that corresponds to a decrease in *ilie* has a positive impact on growth (*ilie* and growth rates vary inversely so life expectancy varies positively with growth rates). The estimation coefficients (table 2) are all statistically significant at 0,001% with the exception of the coefficient for the 0.95 quantile that presents no significance and 0.75 quantile significant at only 0.01%. It is important to bear in mind that adding to statistical significance and apparent different slopes for the estimated quantiles, also the p-value of

equality slope coefficient test indicate that in this case it is rejected the null hypothesis of equal slopes for life expectancy in the growth regression.

**Table 3. Estimates of the quantile panel model and fixed effects model for negative health indicators**

		<i>imr</i>	<i>amr.m</i>	<i>amr.f</i>	<i>under</i>
<b>OLS</b>		-0.4293** (0.1305)	0.0297 (0.0901)	-0.1055 (0.0901)	-0.0400 (0.0589)
<b>Quantile</b>	$\tau = 0.05$	-0.6179*** (0.1761)	-0.0230 (0.0771)	-0.1114 (0.0721)	-0.1143** (0.0427)
	$\tau = 0.25$	-0.5761*** (0.0973)	0.0144 (0.0428)	-0.1427** (0.0485)	-0.0909*** (0.0232)
	$\tau = 0.50$	-0.4637*** (0.1001)	0.0312 (0.0465)	-0.1058* (0.0503)	-0.0538* (0.0246)
	$\tau = 0.75$	-0.3436*** (0.0999)	0.0964* (0.0422)	-0.1457** (0.0503)	0.0029 (0.0304)
	$\tau = 0.95$	-0.1878 (1.1664)	-0.0150 (0.0867)	-0.0150 (0.1093)	0.0227 (0.0597)
<b>Equality slope test</b>		2.1549 (0.0716)	1.9437 (0.1005)	0.9001 (0.4629)	3.7563 (0.0048)**
No. countries		89	90	90	63
Time period		1980-2010	1980-2010	1980-2010	1990-2010

Source: Authors' calculations in R software and edit.

Notes: *imr* - initial level of infant mortality rate; *amr.f* - initial level of adult female survival rate; *amr.m* - initial level of adult male survival rate; *under* - initial level of prevalence of undernourishment. Standard errors in parenthesis. For equality slope test is presented the test's statistic and in parenthesis the p-value. \*\*\*, \*\*, \* and '.' denote the statistical significance at the 0.1%, 1%, 5% and 10% levels, respectively.

In addition to life expectancy, kilocalories per day per person (*ilkal*) is also a positive indicator and since it is introduced in the regression as the inverse it is expected that the estimated coefficient has a negative sign (just like life expectancy). According to the results presented in figure 1 (b) and table 2, the estimated coefficients for this variable confirm the expected negative sign. Figure 1 part (b) suggests that the influence of consumption of calories in the quantiles of growth rates' distribution is quite similar to the results obtained when the regression is estimated by least squares up to the around 0.8 quantile, when the magnitude becomes lower. However, the lowest (0.05) and highest (0.95) quantiles estimated coefficients are not statistically significant and the coefficients for the

other quantiles are very similar which is indeed confirmed by the output of equality slope test.

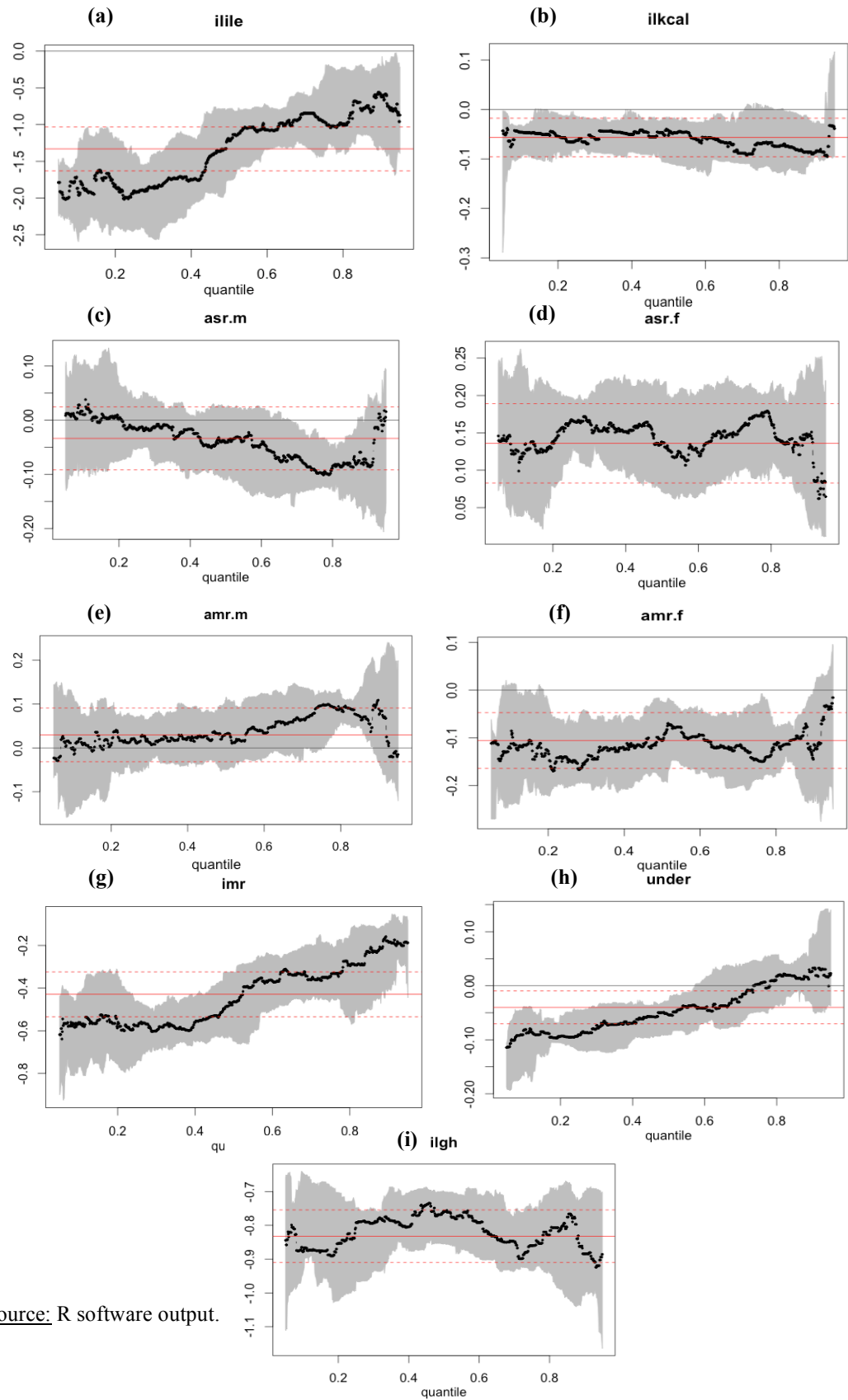
The adult survival rates as well as the adult mortality rates were disaggregated by gender (*amr.m*, *amr.f* and *asr.m*, *asr.f*, m for males and f for females). The results on both indicators differ from the previous positive indicators mostly for male adult rates. Apart from no parameter heterogeneity found by the test on both positive and negative indicators whether male or female, the statistical significance for males is only evident in quantile 0.75. By contrast, the estimators for female variables seem more significant and at more locations of the growth rates distribution. Still regarding female rates, the quantile 0.95 has the lower coefficient but is not significant, like estimators from other health proxies. Also, apparently there is no trend for health coefficients along quantiles, as previously, which we can confirm by looking at figure 1 (d) and (f). For instance, as far as female survival rate is concerned, the correlation is positive for all quantiles but the magnitude of the impact does not show a monotonic behaviour: it increases from the 0.05 to the 0.5 quantiles, it then decreases when we move to the 0.5 quantile to increase again for quantile 0.75 when it reaches its highest value (table 2). This pattern also applies to the estimated coefficients for the female mortality rate (table 3), in absolute values, since the estimated coefficients are always negative as expected. The main difference remains in the significance for levels of growth rates in quantile 0.05 where female adult mortality rates are not significant.

The trend in the behaviour across quantiles of the estimated coefficient for the infant mortality rate (*imr*) is similar to that obtained for life expectancy (see figure 1 (a) and (g)). The statistical significance is identical too: no significance is found for the 0.95 quantile, and the 0.75 quantile coefficient is now even more significant (from 0.01% to 0.001%). The bigger difference in the results produced by infant mortality rate as proxy shows up in the test of parameter heterogeneity for each variable of the model, where the null hypothesis of parameter homogeneity is only rejected at 10% (against 5% for life expectancy slope test). So, at 10% the infant mortality parameters are not homogeneous.

As for the share of undernourishment in population (*under*), the estimated coefficients are negative as expected and some are also statistically significant. And once again it is clear from plot analysis (figure 1 (h)) that running from low quantiles to higher quantiles of growth rates distribution the coefficient magnitude tends to increase. Not only is this tendency observable as the test for equality of coefficients emphasize it rejecting the

hypothesis for coefficients' equality at 0.01%. Though for 0.05 and 0.95 quantiles prevalence of undernourishment is not statistically significant.

**Figure 1. Evolution of health coefficients estimates from quantile regression**



Also confirming theoretical predictions, the estimated coefficients on the inverse of the initial level of public health expenditure per capita (*ilgh*) are negative and statistically significant across all quantiles considered (see table 2). Once more, the slope related to health expenditure is negative because, as previously explained, the equation is estimated with its inverse (*ilgh*). However, regarding the magnitude of the estimated coefficients, although the results for the slope equality test do not reject the null hypothesis of equal slopes, they seem to be slightly higher for median and around 0.8 quantile of the growth rate distribution. From the inspection of the plot (figure 1 part (i)) of the estimated coefficients for the variable related to health expenditure (*ilgh*) we see that the black line, that represents the quantiles' coefficients, does not cross the red dashed line that represents the least squares confidence intervals, thus confirming in part that coefficients do not greatly differ from the least squares estimation. Our results suggest that when countries face public finances sustainability crisis they should cut public expenditure carefully in order not to jeopardize long term output growth, specially for countries like USA where health expenditures are mainly publicly funded. This is in line with the findings of Wang (2011) for the period 1986-2007 who found a positive effect, however with opposite signs for the coefficients of fast growers (positive) and slow growers (negative).

Regarding the control variables, they present similar results in every estimated regression. In table C.1. in annex C we present the estimation coefficients of the quantile regression as well the within least squares model considering life expectancy to proxy health. The equality slope test revealed parameter heterogeneity in education (*educ*), confirmed already by previous studies (Miles 2004). The coefficients are statistically significant and the sign is positive as expected. The variable controlling for convergence (*ly*) also presents the expected sign (negative) and is statistically significant across quantiles. Also statistically significant and with the expected sign in all quantiles is the variable which measures the countries' openness. Population (*n*) in spite of being statistically significant at 0.01% in within model estimation, is only significant in quantile regression estimation at the 0.05 and 0.25 quantiles (at 10%). Government consumption (*g*) on the other hand is not statistically significant in any quantile. For gross fixed capital formation (*gfcf*), the sign is negative which could be explained by non productive investments in that period of time that hence could have led to a crowding-out effect having negative consequences on the economy's performance.

In overall terms, it is observed for the period under analysis that enhancing countries' output per capita long run growth rates could be done by increasing the level of their population's health. These findings are evident through every health proxy used for regressing growth either with positive or negative indicators. The coefficients for the 0.95 quantile are not statistically significant (except for health public expenditure). However, they differ in certain aspects. An observable effect for this period, relates to female health that particularly shows to have influence on economic performance. This could be related to the increasing participation share of female in the labour market in the last decade leading therefore to the influence of health in this period to be mirrored into women's health changes influence. Or even it could be indicating that the economic gains from female's health are higher than from males'. Related to possible females' health externalities on others, namely infants that hence contribute more to foster economic growth<sup>16</sup>. Nevertheless, the magnitude trend through levels of growth performance is unclear, thus requiring a more exhaustive investigation in order to make inferences about it.

The most outstanding issue particularly concern life expectancy, infant mortality rates and undernourishment results, which unfold a rather interesting correlation between the level of quantiles of growth rates and the initial level of health. To date, studies' estimates on the subject have been only considering the impact of health on the average value of growth rates, ignoring the different effects of regressions on the different values of the dependent variable. In other words, ignoring that the influence of health can be heterogeneous depending on the evolution of the economy itself. Our analysis proves this heterogeneity. The output from equality slope's test and the coefficient values show different positive influence of better health according to growth rate of real GDP per capita distribution location. Changes in population's health have greater impact on countries whose growth rates are low (0.05 and 0.25 quantiles), comparing to those performing above the median growth rate (0.75). Therefore, slow growers benefit more from an increase in health than faster growers, once more stressing the importance to carefully take into consideration health investments in periods of economic slackening.

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<sup>16</sup> Prominence for mothers' role is found in Knowles et al. (2002) for education. The idea is developed with an African proverb in the beginning of the paper trying to emphasize that while educating a man is the same as educating one person, educating when we educate a woman we are educating a whole community.

## 6. Conclusion

Recent decades have witnessed huge improvements in vaccination, infectious diseases treatments and access to medical care throughout the world. Nowadays, people are able to live longer than ever expected, and with better quality of life. In this work we revisit the role of health human capital on economic growth by applying a quantile regression approach that allow us to identify different signs and magnitudes for the influence of various health proxies across the distribution of the growth rate of output. This can lead to more specific policy implications regarding health determinants of economic growth, in particular in what concerns health funding by the governments according to economic growth performance. For this purpose, we considered a sample of 92 countries over the period 1980-2010 and applied the quantile techniques proposed by Canay (2011) that allows us to apply quantile regression to panel data.

Our findings suggest that the location on the output growth rate distribution matters in terms of the magnitude of the influence of health on macroeconomic performance in the long run. The evidence that we present considering different health human capital proxies endorses investing in health as a means of improving the growth performance of the 92 countries in our sample. However, countries will benefit more from health investments when they are undergoing periods of growth slowdowns, that is health variables impact is higher for countries performing in the lower quantiles of the growth rate distribution. Also, it stands out the multifaceted aspects of health. Firstly, infants' as well as adults' health status have a prominent effect in explaining income per capita growth. On the other hand, despite some uncertainty on how it works, there is evidence supporting distinction in the contribution of a mother's health from the males'.

In summary, we found evidence of parameter heterogeneity regarding health variables across quantiles in the sense that the size of the parameters of the health variables found to be statistically significant across quantiles with the expected sign, life expectancy, infant mortality rate and prevalence of undernourishment varies across quantiles. These results lead to different policy implications for over-achieving versus under-performing countries. For under achievers (those located at the higher growth quantiles), over the period under analysis better health status predicts higher economic growth. For policies concerns, this represents an alert for countries undergoing slow growth because investing in health fosters growth. Even if those slow growers have enormous levels of health and hardly expect to increase for

example their life expectancy to 100 years, it is important for them not to overlook health improvements because neglecting this can very well mean negative repercussions on long run growth rates. So, especially in times of fiscal constraints health should be an element carefully considered.

We view our results as a starting point to further studies that shed some light on the mixed answers provided so far. While we have shown that there is evidence of parameter heterogeneity in the health-growth relationship in our sample over the period under analysis, further research is needed to understand why such parameter heterogeneity exists. Although quantile regressions identify differences between the behaviour of successful vs. less successful countries, they do not address the question of why some have been more successful than others.

Additionally, future research should explore other mechanisms of transmission from health to economic growth beyond its direct impact on labour productivity in order to provide a more complete picture in terms of direct and indirect effects. Forthcoming research should also focus on developing consistently over time, for a broad variety of countries, a more complete database containing indicators encompassing health dimensions. This way a more complete knowledge and understanding of possible improvements in health and its repercussions on a country's income level will be provided.

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## Appendix

### Appendix I

From PWT and WDI we collected annual data that we used to assemble the initial level of output per capita, education and health in a 5-year period as well as to calculate the average level for the 5-year period, regarding the rest of the variables (see table B.2).

Nevertheless, data on population growth, prevalence of undernourishment and consumption of calories per person per day were lacking a few observations for the period 1980-2010. With the purpose of having a balanced panel model we applied a manipulation technique to the annual data of these variables with the use of R programming. Country by country, with the use of *sas* R package, we applied the method updated by Levinson-Durbin recursion (Brockwell & Davis 1991, p. 242) to fit to each country an autoregressive model. We chose the order of the process that better fitted the data by minimizing Akaike information criteria (AIC). After selecting the model, the values for the missing observations were estimated and we eventually added them to the data set.

In order to study long-term relations, we then for periods of 5-years calculated with the help of R tools the average growth rates of population and real GDP growth; the average of the share of physical capital, the share of government consumption and the share of trade in GDP. For human capital and health, we used the initial levels for the period.

### Appendix II

With the purpose of eliminating the hypothesis of spurious estimations we apply the Levin-Lin-Chu test (Levin et al. 2002) in GRETL to our panel variables and estimation residuals.

This test assumes cross-sectional independence and a common autoregressive parameter for all panels. Therefore, it suggests the following hypothesis:  $H_0$ : each panel contains a unit root;  $H_A$ : each panel is stationary. Thus, the test does not allow for the possibility that some countries contain unit roots while others do not. If the null hypothesis is rejected, we are able to conclude that it is stationary. This test is appropriate to apply to panel data, however does not suit well, according to Levin et al. (2002), for time periods inferior to 5, which is the case of *ilgh*, *under* and *ilkal* which have 3 and 4 time observations for each country. So, for these variables we were not able to compute the test.

## Annexes

### Annex A

**Table A. 1. Summary of selected empirical studies on the effect of health on economic growth**

Study	Health measure (in logs)	Dependent variable	Data	Estimator	Other covariates	Main result(s) on the growth impact of health
Acemoglu & Johnson (2007)	Life expectancy growth	Per capita output growth	40-year panel, 1940-1980, n=47	2SLS IV (disease mortality and global intervention dates)	Postyear dummy x institutions or postyear dummy x initial log GDP	Negative impact due to Malthusian effect. Health impacts population negatively and population impact economic growth negatively.
Aghion <i>et al.</i> (2011)	Log life expectancy growth, initial level of log life expectancy	Annual per capita output growth rate	40-year cross-country, 1960-2000, n=96 (LMW)	IV (LMW and/or ME) OLS Bayesian	Initial log per capita GDP, democracy index, urbanization rate, population density, openness, log of population, log of population x openness	Positive impact on growth rates. Health facilitates growth through investment in physical capital and fertility rates.
Bhargava <i>et al.</i> (2001)	Adult survival rate, ASR x log(GDP)	Per capita GDP growth rates	25-year panel at 5-year interval, 1965-1990, n=92	IV (Dynamic random effects)	Log GDP lagged 5 years, log fertility lagged 5 years, Log (Investment/GDP), tropics, openness	Positive on low income countries. Negative on high income countries. Threshold in health-growth relation significance reached earlier by low income countries.
Bloom <i>et al.</i> (2004)	Life expectancy	Output growth rate	30-year panel data, at 10-year interval, 1960-1990, n=104	instrument (logs) Nonlinear 2SLS	Barro and Lee (2000) average years of schooling, average work experience, tropical area, governance, investment, active population	Positive and significant through health growth and facilitation of technology innovation and diffusion.

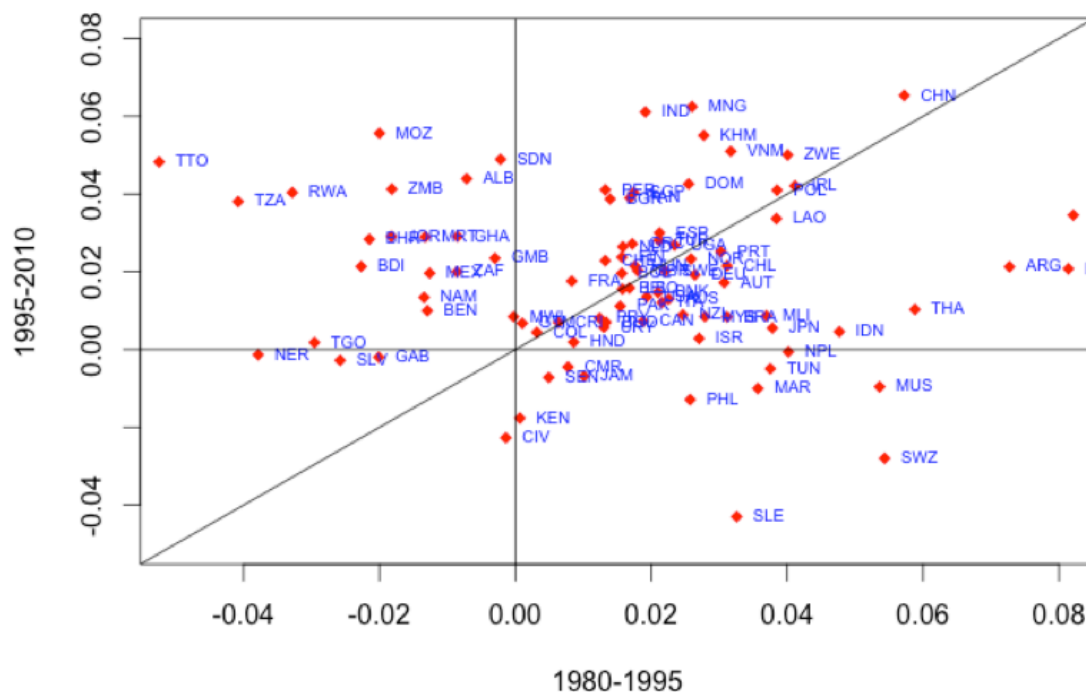
Study	Health measure (in logs)	Dependent variable	Data	Estimator	Other covariates	Main result(s) on the growth impact of health
Cooray (2013)	Life expectancy by gender Survival rates to 65 years by gender (with and without interaction terms with fertility rates, health expenditure per capita and enrolment ratio by gender)	Output growth rate	18-year panel data, 1990-2008, n=210 divided by level of income definition	OLS GMM	Log initial level income per capita, investment in % GDP, trade ratio, fertility rate, monetary aggregate M2, government expenditure ratio, enrolment ratio by gender	Health capital affect economic growth due to health expenditure and education. Positive effect on upper middle and high income countries. Negative on low and low middle income economies.
Eggoh <i>et al.</i> (2015)	Public expenditure on health Life expectancy Survival rate	Output growth rate	14-year panel data, 1996-2010, n=49, African countries	OLS GMM	Log initial level of income, education public expenditure, school enrolment in primary and secondary sector, time dummies, inflation rate, net inflows ratio, money and quasi money ratio, government expenditure ratio (health and education excluded), ratio of exports plus imports	Negative impact of health expenditure. Positive impact of health human capital. Positive health-education interaction. Threshold in health due to education expenditure and vice-versa.
Lorentzen <i>et al.</i> (2008)	Adult and infant mortality rates	Per capita output growth	40-year panel, 1960-2000, n=96	OLS IV (average child and adult mortality rates)	Initial log per capita GDP, Fertility rate, investment share of GDP, secondary school ratio, Sub-Saharan Africa dummy, openness, log population, log population x openness, government consumption in % of GDP	High mortality inhibits growth to escape from poverty trap Physical capital and fertility facilitate health impact on growth. Health-income relation is positive.
Poças (2012)	Life expectancy Infant mortality rate CVA mortality rate Cancer mortality rate Average length of stay Available physicians Acute care beds Health expenditure	Annual average output growth rate	24-year panel data at 5-year interval, 1980-2004, n=22 OECD countries	GMM, simultaneous equations	Log initial level income per capita, investment in % GDP, number of patents, population growth	Positive. More significant health predictors: CVA mortality and number of physicians

Study	Health measure (in logs)	Dependent variable	Data	Estimator	Other covariates	Main result(s) on the growth impact of health
Soukiazis & Cravo (2007)	Life expectancy, infant mortality rate	Average output growth rate	20-year panel data at 5-year interval, 1980-2000, n=77, divided by level of income definition	GMM	(Barro and Lee 2000) average years of schooling, publication ratio of articles published, patents % of population over age 25 ratio, growth rate of working age population, investment % GDP	Positive for low income countries. Insignificant for high income countries.
Wang (2011)	International health expenditure	Annual output growth rate	21-year panel data, 1986-2007, n=31	Quantile regression	-	Lower tail and upper tail show opposite sign. Positive in mean and high upper tail.

Source: Authors.

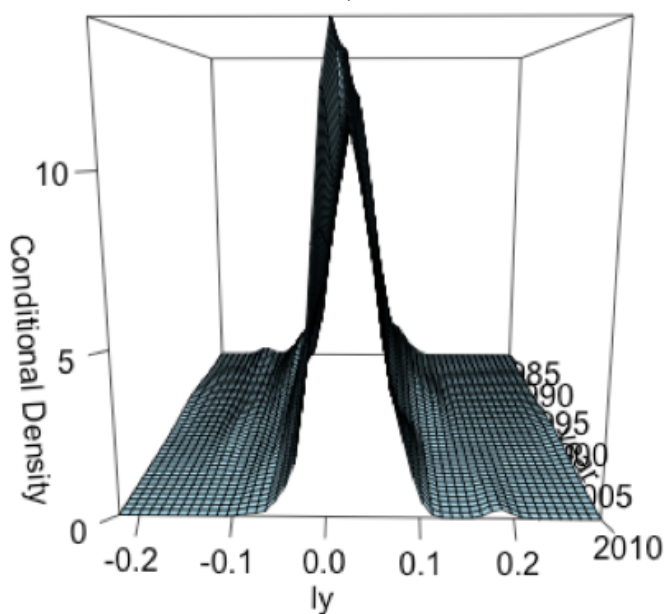
## Annex B

**Figure B. 1. Scatterplot of average GDP per capita growth rates for period 1980-1995 and 1995-2010 (92 countries)**



Source: R software output based on author's calculations.

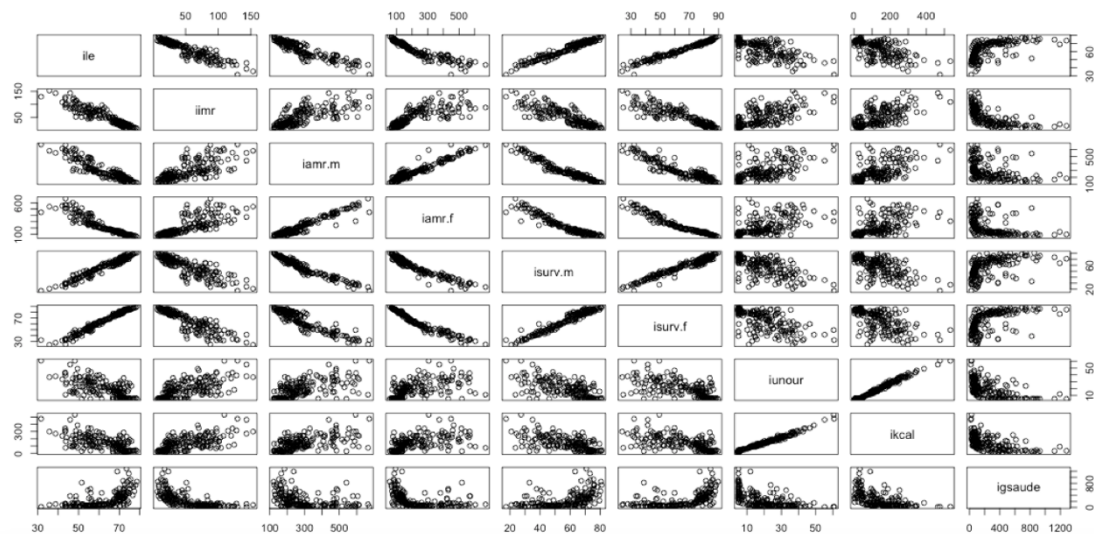
**Figure B. 2. Density function of average per capita growth rate conditioned by the time period (92 countries for 1980-2010)**



Source: R software output.



**Figure B. 3. Matrix of health variables interrelationships (scatter plots)**



Source: R software output.

**Table B. 1. List of countries included in our broader sample's data set**

Countries				
Albania	Costa Rica	Ireland	Namibia	Sudan
Argentina	Cote d'Ivoire	Israel	Nepal	Swaziland
Australia	Denmark	Italy	Netherlands	Sweden
Austria	Dominican	Jamaica	New Zealand	Switzerland
Bahrain	Republic	Japan	Niger	Tanzania
Bangladesh	Egypt	Jordan	Norway	Thailand
Belgium	El Salvador	Kenya	Pakistan	Togo
Benin	Finland	Laos	Panama	Trinidad and
Bolivia	France	Lesotho	Paraguay	Tobago
Botswana	Gabon	Liberia	Peru	Tunisia
Brazil	Gambia	Malawi	Philippines	Turkey
Bulgaria	Germany	Malaysia	Poland	Uganda
Burundi	Ghana	Mali	Portugal	United Kingdom
Cambodia	Greece	Mauritania	Rwanda	United States
Cameroon	Guatemala	Mauritius	Senegal	Uruguay
Canada	Honduras	Mexico	Sierra Leone	Vietnam
Chile	Hungary	Mongolia	Singapore	Zambia
China	India	Morocco	South Africa	Zimbabwe
Colombia	Indonesia	Mozambique	Spain	

Source: Authors' edit.

**Table B. 2. List of variables of our data set**

Notation	Description	Source	Source's notation
$\Delta ly$	Average real GDP per capita growth (calculated the average growth rate of real GDP per capita at chained PPPs divided by total population)	PWT 8.0	rgdpo; pop
$ly$	Initial level of real GDP per capita PPP's logarithm (calculated dividing real GDP in million 2005 USD by total population in millions)	PWT 8.0	rgdpo; pop
$gfcf$	Average share of physical capital's stock in GDP	PWT 8.0	ck
$n$	Population growth rate	World Bank	SP.POP.GROW
$lopen$	Trade (exports plus imports) as a percentage of GDP	PWT 7.1	openk
$g$	Share of government consumption in GDP at current PPP	PWT 8.0	csh_g
$educ$	Barro & Lee (2010) average years of total schooling of people aged over 25	Barro and Lee (2010)	yr_sch
$ilgh$	Public health expenditure per capita (calculated multiplying total health expenditure per capita by public health expenditure as a share of total health)	World Bank	SH.XPD.PCAP.PP.KD SH.XPD.PUBL
$ille$	The inverse of the logarithm of life expectancy at birth in total years' initial level	World Bank	SP.DYN.LE00.IN
$imr$	Initial level of the share of infants dying before reaching one year of age	World Bank	SP.DYN.IMRT.IN
$asr.m$	Initial level of the share of male new-born infants that would survive to age 65	World Bank	SP.DYN.TO65.MA.ZS
$asr.f$	Initial level of the share of female new-born that would survive to age 65	World Bank	SP.DYN.TO65.FE.ZS
$amr.m$	Probability of a 15-year old male dying before reaching age 60 at the beginning of the period	World Bank	SP.DYN.AMRT.MA
$amr.f$	Probability of a 15-year old female dying before reaching age 60 at the beginning of the period	World Bank	SP.DYN.AMRT.FE
$ilkkal$	Inverse of number of calories consumed per day per person's logarithm	World Bank	SN.ITK.DFCT
$under$	Prevalence of undernourishment in population	World Bank	SN.ITK.DEFC.ZS

Source: Databases. Authors' edit.

**Table B. 3. Health variables' correlation matrix (58 countries 1995-2010)**

	<i>ille</i>	<i>ilkal</i>	<i>asr.m</i>	<i>asr.f</i>	<i>amr.m</i>	<i>amr.f</i>	<i>imr</i>	<i>under</i>	<i>ilgh</i>
<i>ille</i>	1.00	-0.58	0.97	0.99	-0.91	-0.97	-0.93	-0.61	0.68
<i>ilkal</i>	-0.58	1.00	-0.58	-0.59	0.56	0.58	0.60	0.99	-0.62
<i>asr.m</i>	0.97	-0.58	1.00	0.96	-0.97	-0.96	-0.86	-0.61	0.59
<i>asr.f</i>	0.99	-0.59	0.96	1.00	-0.91	-0.99	-0.90	-0.61	0.65
<i>amr.m</i>	-0.91	0.56	-0.97	-0.91	1.00	0.93	0.75	0.58	-0.49
<i>amr.f</i>	-0.97	0.58	-0.96	-0.99	0.93	1.00	0.86	0.61	-0.60
<i>imr</i>	-0.93	0.60	-0.86	-0.90	0.75	0.86	1.00	0.64	-0.80
<i>under</i>	-0.61	0.99	-0.61	-0.61	0.58	0.61	0.64	1.00	-0.63
<i>ilgh</i>	0.68	-0.62	0.59	0.65	-0.49	-0.60	-0.80	-0.63	1.00

Source: Authors' calculations with help of R software.

## Annex C

**Table C. 1. Quantile regression estimation results with Life expectancy as the main health related explanatory variable (1980-2010 for 92 countries)**

	<i>FE</i>	<i>Quantile</i>					<i>Equality test</i>
		$\tau = 0.05$	$\tau = 0.25$	$\tau = 0.5$	$\tau = 0.75$	$\tau = 0.95$	
<b>Int.</b>		1.0694*** (0.1203)	1.1161*** (0.1116)	0.9658*** (0.0893)	0.8796*** (0.0876)	0.9196*** (0.1967)	
<i>ille</i>	0.1179*** (0.0312)	-1.7878*** (0.3737)	-1.9006*** (0.3693)	-1.2211*** (0.3199)	-0.9630** (0.3047)	-0.8711 (0.7011)	2.9263 (0.0198)*
<i>ly</i>	-0.0947*** (0.0067)	-0.0975*** (0.0362)	-0.0947*** (0.0030)	-0.0953*** (0.0017)	-0.0921*** (0.0027)	-0.0997*** (0.0061)	1.0509 (0.3793)
<i>educ</i>	0.035*** (0.0019)	0.0157*** (0.0018)	0.0123*** (0.0010)	0.0135*** (0.0008)	0.0118*** (0.0009)	0.0132*** (0.0021)	3.6312 (0.0058)**
<i>n</i>	0.4719** (0.1791)	0.5397. (0.2770)	0.2919. (0.1507)	0.2255 (0.1561)	0.1770 (0.1971)	0.0774 (0.5229)	0.3536 (0.8416)
<i>gfcf</i>	-0.0090*** (0.0020)	-0.0065 (0.0049)	-0.0093*** (0.0015)	-0.0108*** (0.0015)	-0.0096*** (0.0017)	-0.0085* (0.0034)	0.7577 (0.5537)
<i>lopen</i>	0.0225*** (0.0057)	0.0196*** (0.0048)	0.0207*** (0.0026)	0.0218*** (0.0017)	0.0258*** (0.0027)	0.0281*** (0.0048)	1.6778 (0.1522)
<i>g</i>	0.0110 (0.0110)	-0.0283 (0.0362)	-0.0121 (0.0150)	-0.0195 (0.0213)	0.0111 (0.0251)	0.0603 (0.0611)	1.0396 (0.3851)

Source: Authors' calculations with help of R software.

Notes: *ille* - life expectancy; *ly* - initial level of adult male survival rate; *yr\_sch* - initial level of adult female survival rate; *n* - initial level of public health expenditure per capita; *gfcf* - initial level of consumption of calories per day per person; *open* - share of trade in output; *g* - share of government consumption in output; Standard errors in parenthesis. For equality slope test is presented the test's statistic and in parenthesis the p-value. \*\*\*, \*\*, \* and '.' denote the statistical significance at the 0.1%, 1%, 5% and 10% levels, respectively.

